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Air Pollution and Mortality

Results from Santiago, Chile

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The relationship between particulate air pollution and premature death in Santiago, Chile is found to be very similar to results from industrial countries.

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Summary findings

Heavy outdoor pollution is found in developing country cities such as Jakarta, Katowice, Mexico City, and Santiago. But most epidemiological studies of dose-response relationships between particulate air pollution (PM10) and premature deaths are from Western industrial nations. This study of such relationships in developing countries by Ostro, Sanchez, Aranda, and Eskeland fills an important gap. It is also one of the few based on monitored PM10 values, or small particles, which is likely to be a more relevant measure of exposure to air pollution than the more traditional measure of total suspended particulates.

Over several years, daily measures of ambient PM10 were collected in Santiago. Data were collected for all deaths, as well as for deaths for all men, all women, and all people over 64. Deaths from respiratory and cardiovascular disease were recorded separately, and accidental deaths were excluded.

Multiple regression analysis was used to explain mortality, with particular attention to the influence of season and temperature. The association persists after controlling for daily minimum temperature and binary variables indicating temperature extremes, the day of the week, the month, and the year. Additional sensitivity analysis suggests robust relationships.

A change equal to 10-microgram-per-cubic-meter in daily PM10 (about 9 percent) averaged over three days was associated with a 1.1 percent increase in mortality (95 percent confidence interval: 0.6 to 1.5 percent).

Death from respiratory and cardiovascular disease was more responsive to changes in PM10 than total mortality was. The same holds for mortality among men and mortality among individuals older than 64.

The results are surprisingly consistent with results from industrial countries.

This paper—a product of the Public Economics Division, Policy Research Department—is part of a larger effort in the department to analyze environmental policies. A shorter version will be published in *Journal of Exposure Analysis and Environmental Epidemiology*. The study was funded by the Bank's Research Support Budget under the research project "Air Pollution and Health Effects in Santiago, Chile" (RPO 678-48). Copies of this paper are available free from the World Bank, 1818 H Street NW, Washington, DC 20433. Please contact Cynthia Bernardo, room N10-055, extension 37699, or send requests by electronic mail (36 pages). May 1995.

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**Air Pollution and Mortality:
*Results from Santiago, Chile***

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1. Introduction

The need for estimates of environmental benefits in developing countries

Increased attention is now being paid to the need to protect the environment in developing countries, especially after “Our Common Future” (The Report of the Joint U.N. Commission on Environment and Development, 1987) and the Rio Conference on Environment and Sustainable Development. The concern is not only for the loss of natural resources and habitat, but also for health problems associated with pollution (See, for instance, The World Bank’s “World Development Report 1992”, on the environment). Newspaper coverage of health problems in formerly socialist countries, of cholera outbreaks in Latin American and of the Bhopal incident may remind us that problems associated with pollution of air, water and toxics are not limited to western industrialized countries.

For air pollution, the topic of this study, new information about the high concentrations of particulate matter in Eastern Europe and in large metropolitan areas such as Bangkok, Jakarta, Mexico City and Santiago has motivated an examination of the transferability of results from epidemiological studies conducted primarily in the U.S., Canada and Western Europe. There is a strong tradition of cost-benefit analysis in evaluation of projects and policy reform in developing countries, but the inclusion of estimates for environmental effects is rare. Thus, the challenge is posed to provide credible estimates of the benefits that can be provided by pollution reductions in developing countries.

For air pollution, estimates from industrialized countries has emphasized the benefits of improved public health (See, for instance, OECD, 1989)¹. While it is in principle possible to obtain benefit estimates for air pollution reductions directly (say, from survey techniques, or from house values), a more commonly applied approach is to spell out the consequences of reduced air pollution, and then quantify and value these. The present study is a part of a greater effort at the World Bank emphasizing estimation of the health effects of air pollution in developing countries. Ostro (1994) reviewed the literature from developed countries on estimated *dose response functions* - or inference-based studies of associations between ambient air pollution and health impacts, such as premature mortality and respiratory illness. In the review, Ostro concentrated on time-series studies. The review concluded with an application in which effects on morbidity and mortality of several air pollutants were estimated for Jakarta, based on assumptions about transferability of dose response functions. Following Ostro's review, applications of the methodology have been made in many World Bank studies.

While transferability of dose response functions may be a satisfactory approach when locally based inference studies are not available, the present study responds to the need for comparable studies from developing countries. The setting in a developing country may be different in important ways (more time spent out-doors, a younger demographic structure, various aspects of public health status, health services, as well as income related differences,

¹ We limit our attention here to local air pollution problems. The benefits of reduced emissions of climate gases would be global, and hard to estimate, or even model. A recent reference, on the impact on agriculture for the U.S. would be Mendelsohn et al., 1994.

such as nutrition and housing), highlighting the need to enrich the literature with studies from these different settings².

The need for PM10 based studies

In 1986, the U.S. Environmental Protection Agency changed its National Ambient Air Quality Standard for particulate matter from one that included all particles (total suspended particulates or TSP) to one that included only those less than 10 microns in diameter (PM10). Although the standard is based on protecting public health, epidemiologic research on PM10 has been limited by the lack of data from outdoor monitoring stations. In addition, almost all stations that do collect PM10 data operate only every six days, limiting the ability to conduct time-series analysis linking PM10 to various health endpoints, including mortality and morbidity. Thus, in assessing the health effects of particulate matter, many researchers have had to rely on various surrogate measures for PM10. In several recent studies, PM10 data have been available (Pope et al., 1991; Pope et al., 1992; Schwartz, 1993; Dockery et al., 1993), but most have used other pollutant metrics such as TSP (Samet et al., 1981), coefficient of haze (Ostro et al., 1993), fine particulates based on airport visibility (Ostro, 1989), British Smoke (Mazumdar et al., 1982), KM (Shumway et al., 1988) and sulfates (Thurston et al., 1992).

² Regulation of toxic substances is often based on transfer of results from studies of rats under high exposure to assumed effects for humans under low exposure. In this light, the assumption that dose response functions may be transferable from, say people in London to people in Calcutta does seem warranted when local research is not available.

Santiago, Chile

Since 1989, daily measures of ambient PM₁₀ have been collected in Santiago, the capital of Chile. Located in the center of a closed basin, Santiago is about 33 degrees south latitude on the western edge of South America. The population of the metropolitan Santiago area is estimated at 4.4 million, roughly one-third of the entire population of Chile (Ministerio de Economia, 1990). The high ambient levels of particulate matter are a result of emissions from motor vehicles, fossil fuel use for energy production, industrial processes and blowing of resuspended dust, coupled with the unique topoclimatology of the region. Mountains, including the coastal range and the Andes, almost completely surround Santiago. The predominant wind direction is from the southwest - coinciding with the one small gap in the surrounding mountains. The city is situated in a zone with fairly stable atmospheric conditions, including low velocity, turbulence and frequency of winds (Comision Especial de Descontaminacion de la Region Metropolitana, 1990). With prevailing anticyclonic conditions throughout the year, an inversion layer typically exists at between 600 and 900 meters above the city. This layer intensifies in the autumn and winter, preventing natural dispersion of pollutants and trapping most particles within 400 meters above the city (Prendez et al., 1991). Thus, between the months of July and August, during the Chilean winter, particulate concentrations in Santiago are among the highest observed in any urban area in the world (300 to 400 $\mu\text{g}/\text{m}^3$). These particles include a large proportion less than 2.5 microns in diameter, including sulfates (Sandoval and Martinez, 1990). According to a 1985 analysis based on downtown monitors, diesel sources contribute approximately 74 percent of the ambient PM₁₀, with gasoline-powered vehicles, industrial,

residential, and “other” sources responsible for 6, 6, 2, and 12 percent of the concentrations, respectively.³

Outline

Section 2 describes the data, and section 3 presents the methodology. In section 4, we examine the relationship between PM10 and mortality in Santiago. We also report the results of extensive analysis of the sensitivity of the results to alternative regression specifications, methods of controlling for seasonality, functional forms, and health endpoints. Section 5 provides discussion, and Section 6 a comparison with results found elsewhere.

2. Mortality, Air Pollution and Weather Data

For the years 1989 through 1991, daily deaths of residents of metropolitan Santiago were extracted from the mortality records of the Instituto Nacional de Estadísticas. Deaths of Santiago residents that occurred outside of the metropolitan area and deaths from accidents were excluded. In addition to total (all-cause) daily deaths, those due to respiratory disease (ICD 460-519) and cardiovascular disease (ICD 390-448) were tabulated separately. In addition, separate counts of all-cause mortality for males, females, and all people over age 65 were compiled.

Daily 24-hour average concentrations of PM10 were collected from five monitoring sites in Santiago for the same years. Four of the sites are located within a few miles of each other in downtown Santiago, while the fifth is located far to the northeast of the central city. Therefore, as measures of outdoor particles, two alternative metrics were examined: the average of the four downtown monitors and the highest reading from the monitors. In order to examine the spatial

³ Sandoval et al., 1985 - more recent analysis of this kind is not known to be available.

representativeness of the downtown monitors, available daily historical data on total suspended particulates (TSP) from other monitoring sites were obtained for the previous ten years. Comparisons between one of the downtown monitors and five monitors ringing the city indicated daily correlations of 0.68, 0.73, 0.79, 0.85 and 0.92. Since PM₁₀ is likely to be more evenly distributed than TSP, it appears that, at a minimum, the ambient levels move together throughout the basin. Unfortunately, data on PM₁₀ concentrations were not available for every day during this three-year period. Generally, fairly complete data exist for each of the years between April and November, the season of high particulate concentrations. During the rest of the year, missing data are more common. Summary statistics are provided in Table 1. During the three-year period, the mean of the 24-hour average of PM₁₀ concentrations was 115.4 $\mu\text{g}/\text{m}^3$, while the average of the highest daily concentration from any monitor was 141.5 $\mu\text{g}/\text{m}^3$. This compares to U.S. and Chile standards for annual average concentrations of 50 $\mu\text{g}/\text{m}^3$, and a California standard of 30 $\mu\text{g}/\text{m}^3$. The mean PM₁₀ (24-hr average) concentration was 76.2 $\mu\text{g}/\text{m}^3$ during the summer months, and 141.4 $\mu\text{g}/\text{m}^3$ during the winter months. The numbers of observations in 1989, 1990 and 1991 were 267, 277 and 246, respectively.

Table 1. Descriptive Statistics for Air Pollution, Meteorological and Health Variables.

Variable	Mean	Range
PM10 ($\mu\text{g}/\text{m}^3$) ^a (1989)	112.9	32-336
PM10($\mu\text{g}/\text{m}^3$) ^a (1990)	119.5	30-367
PM10($\mu\text{g}/\text{m}^3$) ^a (1991)	113.4	35-308
Maximum PM10 ^b ($\mu\text{g}/\text{m}^3$) (1989)	147.2	35-500
Maximum PM10 ^b ($\mu\text{g}/\text{m}^3$) (1990)	143.5	35-424
Maximum PM10 ^b ($\mu\text{g}/\text{m}^3$) (1991)	132.9	39-340
Ozone (1-hour maximum, ppb)	52.8	11-264
Nitrogen Dioxide (1-hr max, ppb)	55.6	10-258
Sulfur Dioxide (1-hr max, ppb)	59.9	4-363
Minimum Temperature (C ^o)	10.51	-0.23-22.7
Maximum Temperature (C ^o)	22.6	7.37-34.47
Average Daily Humidity	53.1	29.3-93.7
Total Mortality	55.0	22-106
Respiratory Mortality	8.05	0-30
Cardiovascular Mortality	18.0	3-41
Mortality Age 65 and Above	35.6	15-72
Male Mortality	26.8	8-60
Female Mortality	28.1	8-60

a=Daily average of all four monitor readings; b=Daily maximum single monitor reading;

Fairly complete daily data on ozone, sulfur dioxide, and nitrogen dioxide were available during this time period, along with daily data on minimum and maximum temperatures and humidity (Table 1). The correlation coefficients for selected variables are displayed in Table 2.

Particles are positively correlated with oxides of sulfur and nitrogen, and inversely correlated with ozone and temperature.

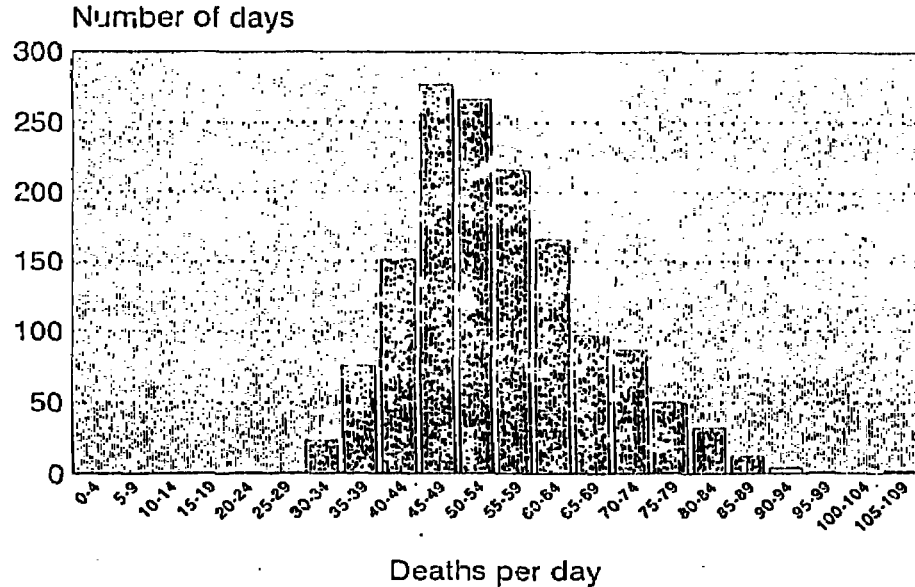
Table 2. Correlation Coefficients for Selected Pollutant and Meteorologic Variables.

	Max PM10	PM10	NO ₂	SO ₂	Ozone	Minimum Temp	Maximum Temp
MaxPM10	1.00						
PM10	0.97	1.00					
NO ₂	0.70	0.73	1.00				
SO ₂	0.65	0.64	0.60	1.00			
Ozone	-0.23	-0.23	-0.06	0.00	1.00		
Minimum Temp	-0.44	-0.45	-0.36	-0.34	0.42	1.00	
Maximum Temp	-0.30	-0.31	-0.15	-0.13	0.68	0.73	1.00

3. Methodology

Mortality counts were first examined to see if they were normally distributed. Figure 1 displays the distribution of counts for total mortality. For these, the normality assumption was not rejected using the Kolmogorov statistic. Therefore, ordinary least squares regression techniques and parametric tests were used in examining the association between air pollution and total mortality. For the other mortality endpoints (subsets of total mortality), the application of a poisson distribution is more appropriate since the normality assumption is less appropriate for events with a lower daily count. For total mortality, to ensure comparability, results using both distributions are reported.

Figure 1: Daily Mortality Santiago, Chile, 1989-91

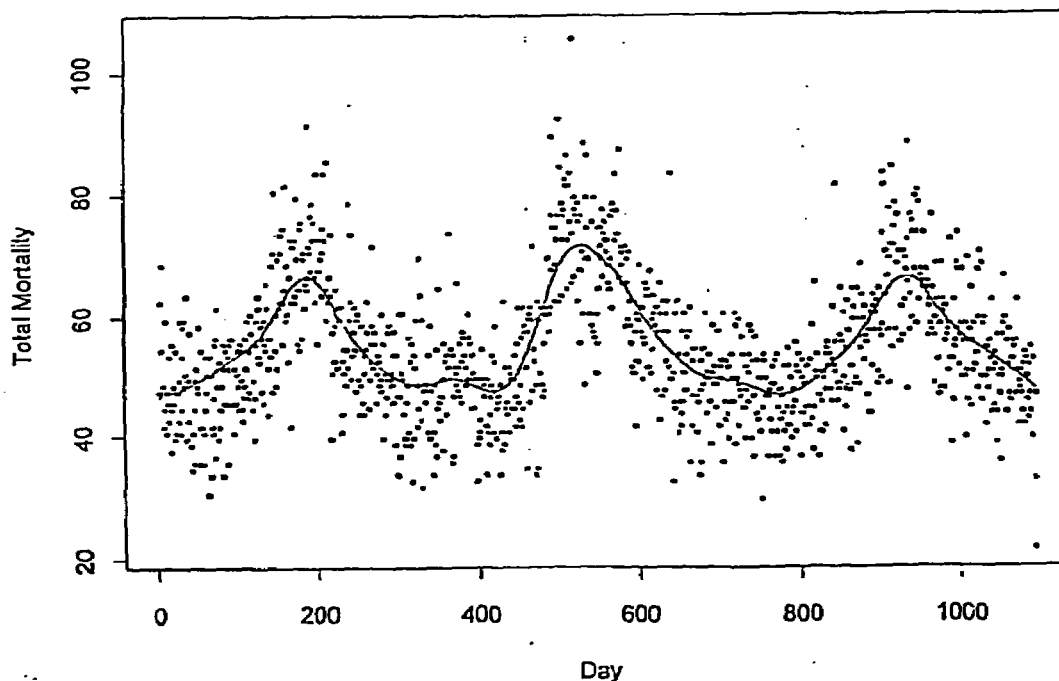


Obviously, air pollution is only one of many factors affecting daily mortality. Therefore, it is important to examine potential confounders, including temperature, month, season, and day of the week. Our analytic approach started with a basic model that included only PM10 as an explanatory variable. We explored both a linear and a semi-log form of PM10, using both the daily mean and the maximum monitor concentrations. Then, in turn, we examined more complex models as additional potential explanatory variables were added to the regression in a hierarchical fashion. At each stage, both the magnitude and significance of the air pollution variables and the additional explanatory power of the new variables were evaluated

Substantial seasonal patterns in mortality exist in Santiago, peaking during the winter months of July and August. Figure 2 displays both the crude mortality counts and a locally weighted, smoothed plot of the seasonal pattern. Control for weather and seasonal cycles in the

model is important, since several recent mortality studies have indicated the influence of temperature on mortality (Kalkstein, 1991). As a result, temperature was first added to the basic model, in terms of minimum, average, and maximum daily levels. Contemporaneous temperature and 1-, 2-, and 3-day lags were individually examined. Temperature was entered as a continuous variable and in binary form to indicate extremes. Thus, days in the lowest or highest 25 percent, 10 percent, 5 percent and 1 percent of either maximum or minimum temperature readings were examined. In the next model specification, season was modeled by use of dichotomous variables for each quarter. Additional controls for season were tested by adding binary variables for month and a time trend was incorporated through a variable indicating the year of study. Day of week was also added to the model with binary variables. The basic model also was rerun for each separate year. Finally, a model was run that included a separate binary variable for each month in each year, a total of 35 additional terms (using the first month for reference), to allow for differences by month within each year.

Figure 2 : Crude, Unadjusted Total Mortality Versus Day of Study



Subsequent to this analysis we considered dose response functions for other mortality endpoints (i.e., subsets of total mortality: respiratory mortality, cardiovascular mortality, mortality for those aged 65 and above, as well as by gender) using a poisson distribution because of the lower mean levels of these counts.

Second, in sensitivity analysis, additional controls for the cyclical nature of the total mortality counts and the effects of temperature were explored through several techniques. The data were stratified first by season and then by year and reanalyzed. Next, the basic regression model was rerun after the coldest 1, 5 and 10 percent of the days were deleted, in turn, to reduce the potentially confounding effect of weather (mortality and particles were both higher during the colder seasons). Another method involved the use of a Fourier series comprising sine and cosine terms as covariates. The series included five terms which incorporate harmonic periods of one year and 6, 4, 3, and 2.4 months. These covariates provide good control for both the short and long waves in the data. The third method to control for cyclical patterns involves the pre-filtering of the data by subtracting multi-day moving averages of 15-days as suggested by previous studies (Kinney and Ozkaynak, 1991). Finally, a generalized additive model was developed using the statistical package S-PLUS (StatSci, 1993) to incorporate non-linear (and non-monotonic) patterns in the temperature-mortality relationship. In this model, the temperature-mortality relationship was smoothed using a nonparametric technique that fits a function in a flexible data-driven manner. Then this function is entered as an explanatory variable in the basic regression model.

In the third sensitivity analysis, other pollutants, including daily maximum 1-hour concentrations of ozone, sulfur dioxide and nitrogen dioxide, were examined with and without PM10 in the model.

In the fourth sensitivity analysis, the effect of extreme or unduly influential observations were examined. Approaches included use of robust regression such as M-estimates and least trimmed squares (LTS) regression (Statsci; 1993). As described by Schwartz (1993), the M-estimation iteratively weights each data point, with increasingly less weight for those observations with larger standardized residuals. The LTS regression model minimizes the sum of the portion of the data with the smallest squared residuals and provides robust estimates with small bias even when the data are highly impacted by outliers. The M-estimates regression reduces the impact of responses that are outliers while the LTS regression reduced the impact of both outlier responses and high leverage points; that is, data points that have different x-values relative to most of the observations.

Finally, in the last sensitivity analysis, the impact of alternative lag structures for PM10 was investigated. Single-period models using PM10 lags of zero to 3 days were run. In addition, models using a 3- and 4-day moving average and a polynomial distributed lag of PM10 were examined.

Since mortality is likely to be serially correlated, autocorrelation corrections were applied to all of the ordinary least squares models using the AUTOREG procedure in SAS.

4. Results

Sensitivity to Specification

Table 3. Regression Results for Total Mortality Using Alternative Model Specifications

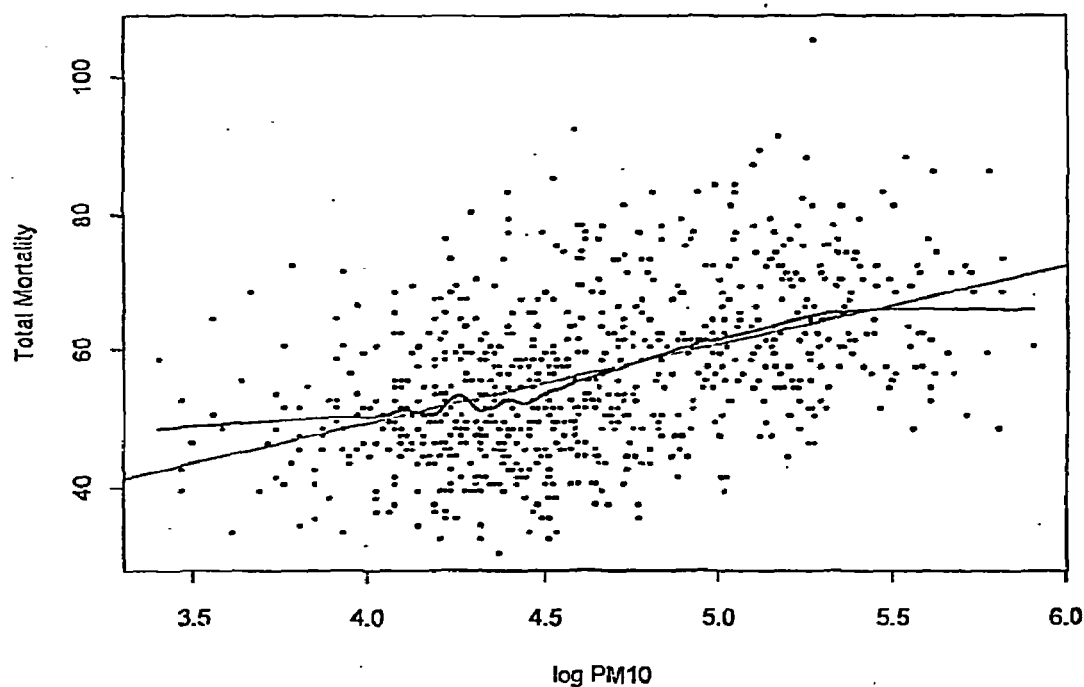
Variables in Model	Beta	s.e.	RR	95%CI
MaxPM10	0.046	0.006	1.13	1.10, 1.16
PM10	0.056	0.007	1.13	1.10, 1.16
Log (maxPM10)	7.82	0.86	1.10	1.06, 1.11
Log (PM10)	7.62)	0.88	1.16	1.14, 1.18
Above +Mintemp (-1)*	5.64	0.78	1.10	1.08, 1.11
Above+Cold10+Hot10	5.62	0.84	1.10	1.08, 1.11
Above+Year	5.78	0.83	1.11	1.09, 1.12
Above+Quarter	4.00	0.92	1.08	1.10, 1.13
Above+Day of Week	6.31	0.84	1.12	1.10, 1.13
Above-Quarter+Month	2.39	0.94	1.05	1.01, 1.08

MaxPM10=daily maximum monitor; PM10=daily average of all monitors,* one-day lag of minimum daily temperature; Cold10=coldest 10 percent of the days; Hot10=hottest 10 percent of the days; Year=binary variable for each year; Quarter=binary variable for each quarter; Month=binary variable for each month. Relative Risk is the ratio of predicted mortality when the pollution variable is set at 1.5 times the mean versus .5 times the mean, respectively. In models with MaxPM10, the mean is of $140 \mu\text{g}/\text{m}^3$ (so the values are 210 vs 70) and in models with PM10 the mean is 115.4. In all models PM10 is significant at $p < 0.0001$ except the last model which is $p < 0.01$. All models were corrected for autocorrelation.

The results of the alternative regression specifications are summarized in Table 3. In a univariate analysis, the association of total mortality with different forms of PM10 were examined, including the mean of the four downtown monitors, the highest daily monitor reading (maximum PM10), and the log of these variables. Each of these terms were strongly associated with mortality ($p < 0.0001$), and explained about 20 percent of the variability in mortality. The log of the maximum PM10 had the strongest association with total mortality although the differences between alternative forms was small. Use of the log of the mean of the daily

concentrations of the four monitors generates an relative risk (RR) associated with a change equal to the mean ($115 \mu\text{g}/\text{m}^3$) of 1.16 ($\text{beta} = 7.62$, $\text{s.e.} = 0.88$) with a 95 percent confidence interval (95%CI) of 1.14-1.18. This suggests that a $10 \mu\text{g}/\text{m}^3$ change in PM10, evaluated at the mean concentration of PM10 is associated with a 1.0 percent change in daily mortality. A correction for serial correlation using a one-day lag effectively reduced the correlation in the residuals so that the null hypothesis of zero autocorrelation could not be rejected. Thus, most of the subsequent analysis used the log of the mean PM10 from the four monitors and a one-period correction for autocorrelation. Figure 3 displays the association of the log of PM10 with mortality, and includes a locally weighted, smoothed plot of the data. Figures A1 through A3 display the association for each year (annex).

Figure 3: Smoothed and Least Squares Fits



Note: Straight line is least squares fit

Of the continuous lagged and unlagged temperature variables (including minimum, maximum or daily average temperature), a one day lag in minimum daily temperature had the strongest association with mortality ($p < 0.0001$). Its inclusion in the regression reduced the estimated coefficient for log PM10 from 7.62 to 5.64 ($RR=1.10$, $95\%CI=1.08-1.11$), but did not affect the statistical significance of the estimate.

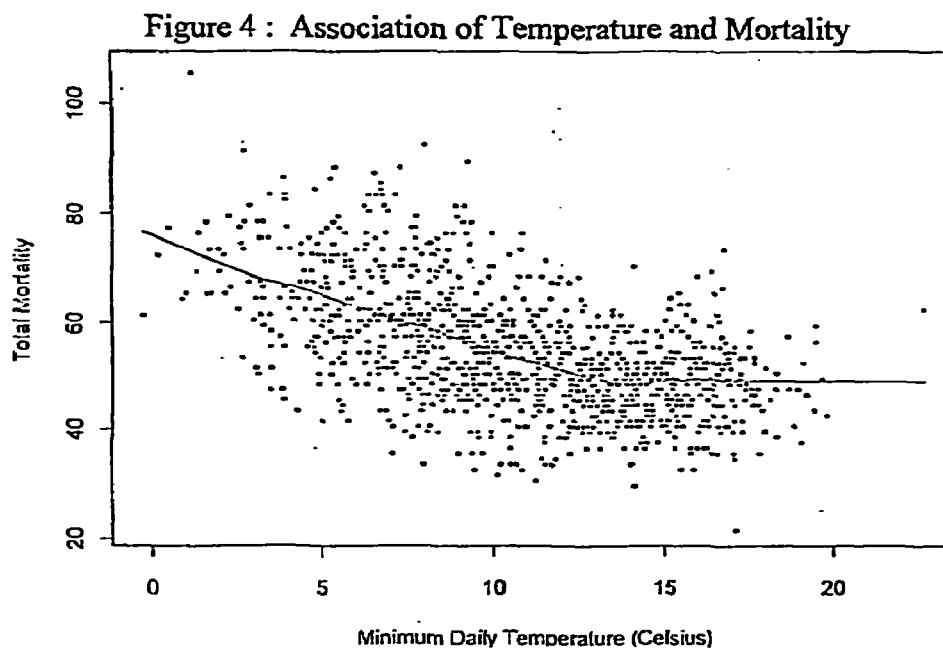


Figure 4 displays the association of temperature and mortality. Note that the non-linear relationship can be fit through use of the generalized additive model. Inclusion of other continuous weather terms, including maximum temperature and different forms of daily humidity, did not further improve the model fit. In examining temperature extremes, binary variables representing the coldest and hottest 10 percent of the days had the strongest association with mortality. The inclusion of these two terms did not affect either the magnitude or statistical significance of the PM10 coefficient. The addition to the model of the year of study and dichotomous variables for each quarter further reduced the magnitude of the log PM10

coefficient to 4.00 (RR=1.08, 95%CI=1.06-1.11) with no change in its statistical significance.

The regression estimates for all the terms in this model are provided in Table 4.

Table 4. Regression Results for Basic Model Using Ordinary Least Squares

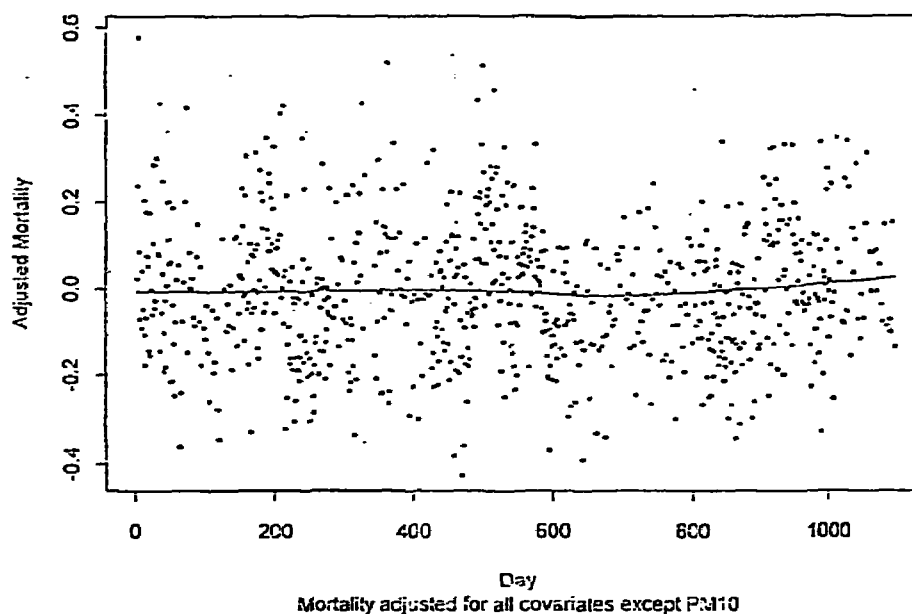
Variable	Beta	s.e.	p-value	Relative Risk
log(PM10)	4.00	0.92	.0001	1.08
Min Temp (one-day lag)	-0.64	0.17	.0001	1.17
Cold10	1.86	1.30	.15	1.05
Hot10	3.11	1.26	.01	1.08
Quarter=2	3.88	0.72	.0001	1.10
Quarter=3	8.74	1.54	.0001	1.23
Quarter=4	1.67	1.26	.19	1.04
Year=2	2.29	0.98	.02	1.06
Year=3	0.90	1.00	.37	1.02
Constant	37.70	5.16	0.0001	--

Note: Cold10=coldest 10 percent of the days; Hot10=hottest 10 percent of the days. Relative risks are calculated for a $115 \mu\text{g}/\text{m}^3$ (the mean) change in PM10 and a 10 degree C decrease in temperature.

A 10 degree Centigrade change in minimum daily temperature generated an RR of 1.17, while the coldest and warmest 10 percent of the days give an RR (relative to the rest of the days) of 1.08 and 1.06, respectively. When day of the week is added to the model, the estimated log PM10 coefficient increases to 6.31 (RR = 1.12). Dropping the binary variables for each quarter and adding one for each month reduced the estimated PM10 effect, but it was still highly significant. This model appears to control well for seasonality. For example, the residuals (see Figure 5), defined as mortality minus the regression estimate based on a Poisson regression

model without PM10 (but including minimum temperature, binary variables indicating the coldest and hottest 10 percent of the days, quarter, and year) no longer exhibit the seasonal pattern observed in Figure 2.

Figure 5: Smoothed Fit of Residuals Using Poisson Model



When a “best” model without PM10 was specified, the entry of log PM10 was significant with results essentially identical to the above. The analysis by year also indicated a significant association between mortality and PM10 in each year. The respective RRs for 1989, 1990 and 1991 were 1.14, 1.11 and 1.06, respectively. The model that included 35 more terms denoting each separate month in the study generated an $RR = 1.04$ (95% C.I. = 1.01-1.06) for PM10. Figure 3, which compares the semi-log model with the data-dependent locally weighted smoothed fit indicated that the semi-log model fits the bulk of the data well.

Alternative mortality endpoints

Poisson models were estimated for cardiac-specific mortality, respiratory-specific mortality, mortality for men, women, and those over 65. These models included log PM10, one-day lag of minimum daily temperature, the hottest and coldest 10 percent of the days, year, and binary variables for quarter (Table 5). The log of PM10 concentrations was statistically significant in all cases. The strongest associations ($p < 0.0001$) were with total mortality, respiratory-specific mortality, and mortality for those above age 65, and for males. The highest RR (1.15) is estimated for respiratory-specific mortality as one might have conjectured. Slightly lower statistical significance was found for the relationship between PM10 and both cardiovascular-specific mortality and female mortality ($p = 0.0005$ and $p = 0.004$, respectively). Of additional interest, all of the other covariates in the model were significant for each of the health endpoints with the exception of the coldest and hottest days for cardiovascular mortality, and the coldest days for female-specific mortality.

Table 5. Poisson Regression Results for Mean PM10 on Alternative Mortality Endpoints

Endpoint	Beta	s.e.	p-value	RR	95%CI
Total	0.075	0.013	<0.0001	1.08	1.06, 1.12
Respiratory	0.127	0.032	<0.0001	1.15	1.08, 1.23
Cardiovascular	0.076	0.022	0.0005	1.09	1.04, 1.14
Male	0.101	0.018	<0.0001	1.11	1.07, 1.14
Female	0.050	0.018	0.004	1.06	1.02, 1.10
Age 65 and above	0.091	0.017	<0.0001	1.11	1.07, 1.14

Regression model includes: log (PM10), minimum temperature (one day lag), binary variables for coldest and hottest 10 percent of the days, and dichotomous variables for year, and quarter, corrected for autocorrelation. Beta and standard error are $\times 100$. Relative risks are calculated for a $115 \mu\text{g}/\text{m}^3$ (the mean) change in PM10.

Seasonality and the Effects of Temperature

Sensitivity analysis was conducted to examine the impacts of PM10 after different adjustments for seasonal effects and temperature were employed. Regressions were rerun after the data were stratified by winter versus summer. In both seasons, PM10 was associated with total mortality, but a stronger association was observed in winter (RR= 1.07, $p = 0.001$) than in summer (RR = 1.06, $p = 0.10$). Consecutively deleting the days with the coldest 1, 5 and 10 percent highest temperatures from the analysis did not markedly alter either the magnitude of the effect or the statistical significance of PM10, with resultant RR = 1.09, 1.09, and 1.11, respectively. When five sine and five cosine terms were added to the full model (to control the cyclical nature of mortality in a different manner) the effect of PM10 changed slightly (RR= 1.04).

Finally, when the general additive model was used to explicitly incorporate the influence of the nonlinear effect of temperature, the RR = 1.09 is very similar to that of previous models. The results of these models are summarized in Table 6.

Table 6. Relative Risks for PM10 Using Alternative Regression Models.

Model	Relative Risk
1. Ordinary Least Squares (OLS)	1.08
2. Poisson	1.08
3. Respiratory-specific	1.15
4. OLS using 3-day moving average	1.12
5. Ordinary Least Squares with trig terms	1.04
6. M-estimation	1.09
7. Least Trimmed Squares	1.09
8. Generalized Additive Model	1.09

- 1: Includes log (PM10), minimum daily temperature (lagged one day), binary variables for the coldest and hottest 10 percent of the days, and binary variables for quarter and year.
- 2: Same specification as above, using Poisson model.
- 3: Same specification as above.
- 4: Same specification as #1 using 3-day moving average of log (PM10).
- 5: Same specification as #1 plus 5 sine and 5 cosine with harmonic phases of 1 year to 2.4 months.
- 6: Same specification as #1 using robust estimation technique that reduces influence of observations with varied outlier response.
- 7: Same specification as #1 using robust estimation technique that reduces influence of observations with varied outlier response and observations with high leverage.
- 8: Same specification as #1 using model incorporating nonparametric smooth of daily minimum temperature.

NOTE: Relative risks calculated at the mean for a change of 115 $\mu\text{g}/\text{m}^3$.

Other pollutants

The effects of other pollutants were examined by considering each alone and with PM10 in the regression model. The model with the additional 35 binary variables signifying the month of the study was used since it was likely to provide good control for seasonality. However, the results were robust to alternative models and functional forms. When considered alone, the 1-

hour maxima of sulfur dioxide and nitrogen dioxide were associated with total mortality (RR= 1.01; 95% C.I. = 1.00- 1.03 and RR= 1.02; 95% C.I. 1.01-1.04, respectively), but ozone was not (RR=0.97, 95% C.I. = 0.96-0.98). However, when PM10 was added back into the model, none of the other pollutants was statistically significant. The respective RRs for sulfur dioxide, nitrogen dioxide and ozone were 1.01 (95%C.I = 0.98-1.03), 0.99 (0.97-1.01), and 0.98 (95%C.I. = 0.96-1.00). The magnitude and significance of PM10 was basically unchanged regardless of whether the other pollutants were in the model.

Influential Observations

In this analysis, two robust estimation techniques were used to reduce the impact of influential observations. Again the results did not change significantly; both the M-estimate and the LTS regression produced RRs of 1.09. Results for these alternative models are summarized in Table 6.

Lag structure

Examination of various period lags for PM10 indicated that contemporaneous exposures and 1-, 2-, or 3-day lags were all associated with mortality. A model using a polynomial distributed lag that endogeneously determines the weights of the lags suggests that mortality is associated with exposures with 0-, 1-, and 2-day lags. Likewise, a moving average of either 3 or 4 days generated a good fit to the data. The amount of explained variation (R^2) of the models with alternative lag specifications was fairly similar, generally between 0.40 and 0.44. These estimates may be slightly biased because more missing values occurred during the lower

pollution days in the warm season. Table 7 summarizes these results. As an example of how to interpret the results, the 6.96 coefficient of log PM-10 in the 3-day model implies that mortality increases by 1.1% for each $10\mu\text{g}/\text{m}^3$, with a confidence interval of .6 to 1.5 % (mean PM10 is 115, and mean mortality is 55).

**Table 7. Effects of Alternative Lags of Log PM10 on Total Mortality Counts
(Estimated Regression Betas with t-statistics in parentheses)**

PM10 Lag Structure	Single Period Model				Moving Average	Polynomial Distributed Lag
PM(10)	3.79 (4.11)	-	-	-		2.60 (2.75)
PM(t-1)	-	2.16 (2.84)	-	-		2.10 (4.05)
PM(t-2)	-		4.09 (4.56)	-		1.61 (3.30)
PM(t-3)				2.09 (2.27)		1.12 (1.24)
3-day average					6.96 (4.44)	
4-day average					7.33 (4.34)	

Regression covariates include log (PM10), lag of minimum daily temperature, cold10, hot10, and binary variables for quarter and year.

5. Discussion

This analysis demonstrates associations between PM10 and daily mortality in Santiago, Chile. This association was sustained after control for several potential confounders, including temperature, season, month, and day of week. Special attention was given to reducing the confounding that may occur because of the long wavelength patterns in mortality and air

pollution; both of which peak in the winter. Dropping the days with the lowest temperatures did not alter the results. As shown in Figure 4, although lower temperatures are associated with increased mortality, the highest mortality days are not necessarily the coldest. Temperature appears to have a significant independent effect on mortality: a 10 degree Centigrade decrease is associated with an RR of 1.27. However, the analysis suggests that an air pollution effect persists, independent of temperature and season. This was confirmed in several different ways. First, after stratifying by season and considering the winter period alone (when ozone concentrations were very low and the long-wave patterns of mortality and PM10 were minimized), a significant association between PM10 and mortality persists. Second, the plot of adjusted mortality in Figure 5 indicates that the basic model effectively minimized the seasonal effects on mortality. Finally, even after the coldest 1, 5 and 10 percent of the days were eliminated from the analysis, an association between PM10 and mortality remained.

Analyses for individual years indicate that the association is robust. Smoothed fits for two of the years indicate a stronger response to PM10 beginning at approximately $90 \mu\text{g}/\text{m}^3$, while the fit for the third year (1989) does not appear to indicate a threshold. The alternative mortality endpoints were all related to PM10, with respiratory-specific cases having the highest RR. The association of air pollution and mortality is strongest among the groups expected to be at higher risk: the older subpopulation and men.

When other pollutants (sulfur dioxide, nitrogen dioxide, and ozone) were considered in the model, they did not appear to have an independent effect on mortality. In addition, the magnitude and the significance of the coefficient for PM10 was generally unchanged by their

inclusion. Examination of the lag structure indicated that mortality was associated with contemporaneous exposure as well as with lags of one or two days.

6. Comparison with other studies

Since this is one of the few epidemiologic studies of air pollution for a developing country, it is of interest to compare the quantitative implication of these findings with those from the developed world. Recent reviews of the mortality studies undertaken in cities such as London (England), Steubenville (Ohio), Detroit, Philadelphia, Birmingham (Alabama), Santa Clara County (California), and the Utah Valley (Ostro, 1993; Dockery and Pope, 1994) suggest that a $10 \mu\text{g}/\text{m}^3$ change in PM₁₀ is associated with approximately a 1 percent change in mortality, while a recent meta-analysis (Schwartz, 1994) suggests a range of 0.7 to 1.1 percent. This magnitude appears to exist over a wide range of climates, populations, and variation of chemical constituents of PM₁₀.

The results for Santiago are fairly consistent with these other analyses. For example, using the basic semilogarithmic model (Table 4), a $10 \mu\text{g}/\text{m}^3$ change around the mean of $115 \mu\text{g}/\text{m}^3$ in PM₁₀ corresponds to a 0.6 percent change in mortality, with a 95% confidence interval of 0.4 to 0.7 percent. The poisson model (Table 5) suggests a mortality increase of 0.7 percent at the mean concentration, and increases of 1.4 percent and 0.4 percent when evaluated at 50 and $150 \mu\text{g}/\text{m}^3$, respectively. Finally, the model using the 3-day moving average suggests a 1.1 percent change at the mean concentration. These estimates are well within the range of the results reported in the U.S. Table 8 provides a comparison with time series studies from the U.S, and one from China.

Table 8. Time Series Models of Total Daily Mortality

Study ^d	Mortality Coefficient ^b : Percent/10 $\mu\text{g}/\text{m}^3$	Pollutant Measure Used	Mean PM10 $\mu\text{g}/\text{m}^3$ (converted)
Philadelphia	1.3% (.7,1.8)	2-day TSP	42 ^a
Detroit	1.1% (.5,1.6)	1-day TSP	48 ^a
Utah Valley	1.6% (.9,2.3)	5-day PM10	47
Birmingham, Alabama	1.1% (.2,2)	3-day PM10	48
Steubenville, Ohio	.7% (.4,9)	1-day TSP	62 ^a
Beijing, China	.7% ^c (.05, 1.4)	1-day TSP	206 ^a
Santiago, Chile	.7% (.5,1)	1-day PM10	48
Santiago, Chile	1.1% (.6,1.5)	3-day PM10	48

^a A conversion, assuming .55 grams PM10/gram TSP is used for studies in which TSP was the original measure.

^b 95 percent confidence interval in parenthesis. ^c The coefficients is from "Summer only" model. In a full-year model, the coefficient for TSP was not significantly different from zero for total mortality (SOx was, and TSP was for Chronic Obstructive Pulmonary Disease Mortality, See table 9). ^d The studies are, respectively: Schwartz et al. (1992), Schwartz (1991), Pope et al. (1992), Schwartz (1993), Schwartz et al. (1992), this study, Xu et al. (1994).

Recently, there has been published two studies from developing countries. Xu et al (1994) finds total mortality to be significantly associated with TSP in Beijing in a model for the summer months, but the association is not significant in a full-year model. A 10 microgram change in PM-10 was associated with a .7% increase in total mortality in the summer-month model, using a standard conversion to PM10 (confidence interval of .3%, 1%, Table 8)⁴. Saldiva et al (1994) studied the effect of PM10 on total mortality among those above 65 years of age in

⁴ Mortality due to chronic obstructive pulmonary disease was significantly associated with TSP in a full year model (Table 9). Sox was also found to be a significant determinant of mortality.

Sao Paulo, Brazil. A 10 microgram change in PM10 was associated with a 1.3% increase in mortality (.71%, 1.92%), with PM10 expressed as a 2-day moving average (Table 9).

Analysis by alternative subgroups (by gender, by age group) and by diagnosis is reported in some of these studies. In Table 9, such results are compared for three U.S. studies, for Sao Paulo, Beijing and for Santiago. The studies have in common that mortality among the elderly, and due to diseases of the heart and lungs are more responsive to changes in particulate air pollution than is total mortality. The higher effect among men is also found in Dockery et al 1993b, even after including the effects of smoking and work exposure. These and other estimates for subgroups indicate a route towards understanding more of the underlying phenomena, and in particular for differences among studies, to improve the precision in transfers of dose-response functions. In particular, if transferred dose response functions for total mortality are thought of as sums of those for age-groups and by diagnoses, then details on age structure and disease patterns can improve upon the transfers.

Between Santiago, Chile, and cities of the U.S. there are important differences in demographics, behavior (e.g., time spent outdoors), smoking habits, competing risks, and many factors related to income - nutritutional status, occupational exposure, the quality of health care, housing and supporting infrastructure. For example, regarding demographics, the infant mortality rate in Chile is 17 per 1,000 versus 9 per 1,000 in the United States. In addition, 6 percent of Chile's population is at least 65 years old, versus 12.9 percent in the United States. Finally, the crude death rate in Chile is only 2/3 of that in the United States. Thus, one may perhaps be surprised that the Santiago results are so much in line with the U.S. experience.

Table 9. Alternative mortality subgroups, comparisons across studies

Study ^a	Category	Coefficient for 10 μ g/m ³	Percentage of Deaths
Utah Valley	Total Mortality	1.6 %	100
	Respiratory Disease	4.3 %	10
	Cardiovascular Disease	2.0 %	46
	All Other	0.5 %	44
Birmingham, Alabama	Total Mortality	1.1 %	100
	Chronic Lung Disease	1.6 %	n.a.
	Cardiovascular Disease	1.7 %	n.a.
	Other	0.6 %	n.a.
Philadelphia	Total Mortality	1.3 %	100
	Chronic Obstructive Pulmonary D.	3.4 %	2
	Pneumonia	2.0 %	3
	Cardiovascular Disease	1.8 %	46
	Total, over 65 years	1.8 %	65
Beijing	Total (summer only)	0.7 %	
	Chr.Obstr.Pulmonary D.	1.8 %	3
Sao Paulo	Total, over 65 years	1.3%	n.a.
Santiago	Total Mortality	0.7 %	100
	Respiratory Disease	1.3 %	15
	Cardiovascular Disease	0.8 %	33
	Male	1.0 %	49
	Female	0.5 %	51
	Total, over 65 years	1.0 %	55

a: Studies are cited in Table 8.

Other possible reasons for any differences in the estimates in Santiago relate to the use of the downtown monitors. Most of the studies in the U.S. have averaged readings from monitors located throughout the metropolitan area under study. The single downtown monitor (or the average of four adjacent downtown monitors) likely overestimates the actual exposure (and

variation in exposure) for a majority of the metropolitan Santiago population. As a consequence, for a given impact of pollution on mortality, if the variation in PM10 levels are overestimated, then the regression coefficient is likely to be underestimated.

The above results indicate that it is reasonable to conclude that the estimated impact of PM10 on mortality in Santiago is consistent with that found in other studies in the United States. Based on these findings, it appears that it is justifiable to extrapolate the results from the U.S. to other, less-developed countries whenever one cannot undertake local research. However, for countries with other characteristics than Chile in such factors as time spent outside, baseline health status, and medical care and access, such transfers should be applied with caution. Data sets from other settings are now becoming available, and should be used to enrich our understanding, for specific as well as general purposes.

In terms of future research, ongoing institutional and technical developments in many cities around the world indicate that promising data sets will be forthcoming. Carefully managed and undisturbed data collection, emphasizing daily observation of pollution and weather data, will be essential. Then, these data should be made available for researchers with data on mortality and illness with a similar time series structure⁵.

Finally, time series studies like this one may fail to capture important facets of the health effects of air pollution. First, if there are additional longer term effects, such as cumulative contribution to respiratory mortality and certain types of cancer, then they may remain undetected in approaches based on inference from shorter term co-variation. Secondly, these

⁵ Aranda et al (1995) combines the Santiago pollution data with data on children's respiratory illness.

results on premature mortality provide little indication of *how premature* the incidences are (a question that may be relevant when valuation of health effects is asked for⁶). Thus, studies with other types of data and methodologies will be useful as well. Two very different examples may be mentioned: Gil et al. (1993) analyses the particulates found in the air in Santiago, to indicate high levels of mutagenic and carcinogenic compounds. Dockery et al., 1993b (from the Harvard Six Cities Study), uses a panel of 8000 individuals in 6 cities over 15 years, to show strong longer term effects. These and other studies indicate that the reduced premature mortality offered by air quality improvements would not be limited to short term variations in the timing of death for frail individuals, but would include healthy life years saved for many.

⁶ See World Bank, 1994, for cost benefit analysis of an air pollution control program for Santiago.

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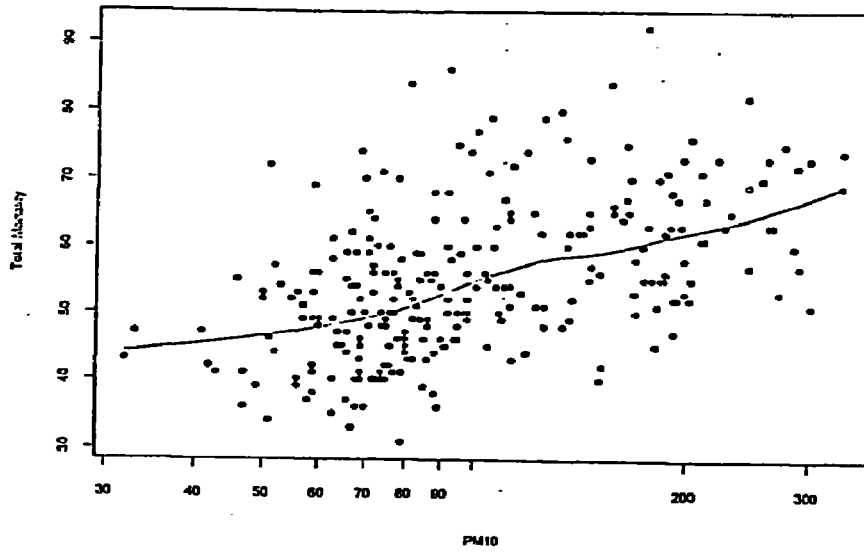
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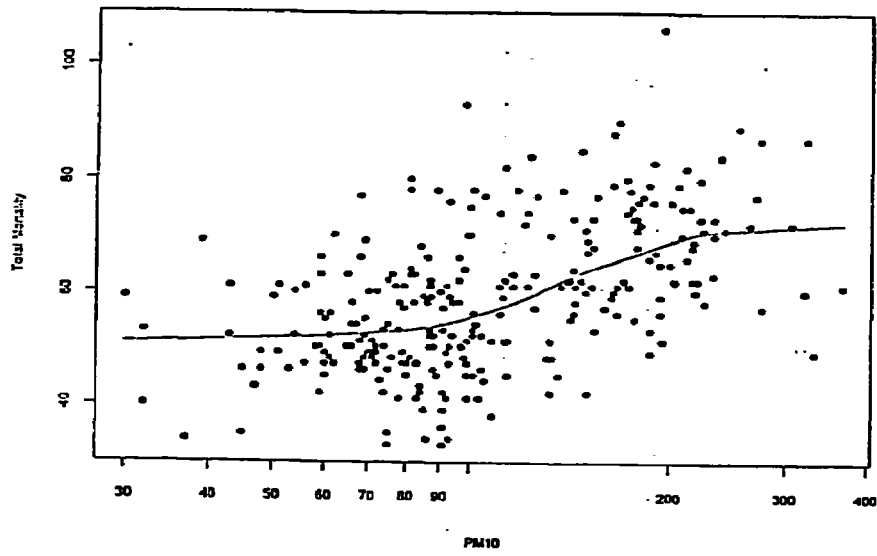
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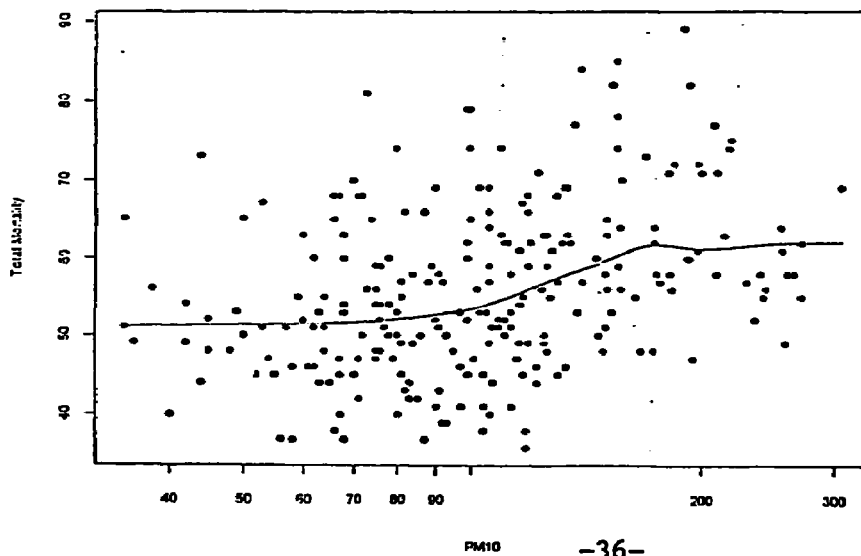
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